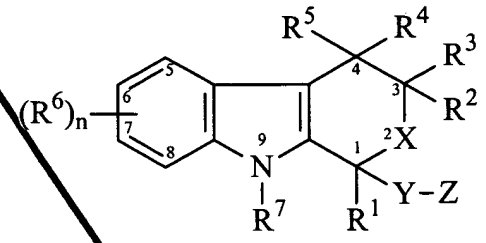
What is claimed is:

1. A compound of formula (I):



(I)

wherein R^1 is lower alkyl, lower alkenyl, (hydroxy)lower alkyl, lower alkynyl, phenyl, benzyl or 2-thienyl, R^2, R^3, R^4 and R^5 are the same or different and are each hydrogen or lower alkyl; each R^6 is individually hydrogen, lower alkyl, hydroxy, (hydroxy)lower alkyl, lower alkoxy, benzyloxy, lower alkanoyloxy, nitro or halo, n is 1-3, R^7 is hydrogen, lower alkyl or lower alkenyl, X is oxy and thio, Y is carbonyl, $(CH_2)_{1-3}$, $(CH_2)_{1-3}SO_2$ or $(CH_2)_{1-3}C(O)$, and Z is (ω-(4-pyridyl)(C_2-C_4 alkoxy), (ω-((R^8)(R^9) amino)(C_2-C_4 alkoxy), wherein R^8 and R^9 are each H, (C_1-C_3)alkyl or together with N are a 5- or 6-membered heterocyclic ring comprising 1-3 N(R^8), S or nonperoxide O; an amino acid ester of (ω-(HO)(C_2-C_4))alkoxy, N(R^8)CH(R^8)CO_2H, 1′-D-glucuronyloxy; or Y-Z is $(CH_2)_{1-3}R^8$; wherein R^8 is OH, (C_2-C_4)acyloxy, SO_3H, PO_4H_2, N(NO)(OH), SO_2NH_2, PO(OH)(NH_2), or tetrazolyl; or a pharmaceutically acceptable salt thereof.

2. The compound of claim 1 wherein Z is the L-valine or L-glycine ester of 2-hydroxyethoxy.

3. The compound of claim 1 wherein Z is N-morpholinoethoxy.

4. The compound of claim 1 wherein each R^8 is H, CH_3 or i-Pr.

5. The compound of claim 1 wherein Z is $OCH_2CH_2N(CH_3)_3^+$.

6. A composition comprising the compound of claim 1 in combination with a pharmaceutically acceptable carrier.

7. The composition of claim 6 which is a tablet, granule or capsule.

8. The composition of claim 6 wherein the carrier is an aqueous vehicle.

9. The composition of claim 8 which is an aqueous solution.

10. A method of inhibiting cancer comprising administering an effective amount of the compound of claim 1 to a mammal afflicted with cancer.

11. A method of inhibiting cancer comprising administering an effective amount of the composition of claim 6 to a mammal afflicted with cancer.

12. The method of claim 10 or 11 wherein the cancer is prostate cancer.

13. The method of claim 10 or 11 wherein the cancer is multiple myeloma.

14. The method of claim 10 or 11 wherein the cancer is chronic lymphocytic leukemia.

15. The method of claim 11 wherein the composition is administered orally.

16. The method of claim 15 wherein an enterically coated dosage form is administered.

17. The method of claim 11 wherein the composition is administered parenterally.

18. The method of claim 11 wherein the composition is administered in combination with a chemotherapeutic agent.

19. The method of claim 12 wherein the composition is administered in combination with a chemotherapeutic agent.

20. The method of claim 18 wherein the chemotherapeutic agent is mitoxanthrone, prednisone, estramustine, vinblastine or a combination thereof.

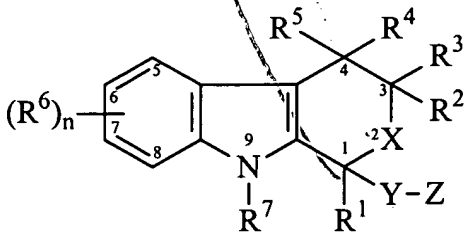
21. The method of claim 12 wherein the chemotherapeutic agent is an anti-androgen.

22. The method of claim 21 wherein the anti-androgen is bicafutamide, nilutamide, flutamide, cycloproterone acetate or a combination thereof.

23. The method of claim 21 wherein the anti-androgen is leuprolide acetate, goserelin acetate or a combination thereof.

24. A therapeutic method comprising:

(a) evaluating the level of at least one of PPAR-γ, Mcl-1 or Bag-1 in cancer cells isolated from a patient afflicted with prostate cancer to determine if said level is sufficiently high so that said cells would be susceptible to inhibition by a compound of formula (I):



(I)

wherein R^1 is lower alkyl, lower alkenyl, (hydroxy)lower alkyl, lower alkynyl, phenyl, benzyl or 2-thienyl, R^2, R^3, R^4 and R^5 are the same or different and are each hydrogen or lower alkyl; each R^6 is individually hydrogen, lower alkyl, hydroxy, (hydroxy)lower alkyl, lower alkoxy, benzyloxy, lower alkanoyloxy, nitro or halo, n is 1-3, R^7 is hydrogen, lower alkyl or lower alkenyl, X is oxy and thio, Y is carbonyl, $(CH_2)_{1-3}$, $(CH_2)_{1-3}SO_2$ or $(CH_2)_{1-3}C(O)$, and Z is (ω-(4-pyridyl)(C_2-C_4 alkoxy), (ω-((R^8)(R^9) amino)(C_2-C_4 alkoxy), wherein R^8 and R^9 are each H, (C_1-C_3)alkyl or together with N are a 5- or 6-membered heterocyclic ring comprising 1-3 N(R^8), S or nonperoxide O; an amino acid ester of (ω-(HO)(C_2-C_4))alkoxy, N(R^8)CH(R^8)CO_2H, 1′-D-glucuronyloxy; or Y-Z is $(CH_2)_{1-3}R^8$ wherein R^8 is OH, (C_2-C_4)acyloxy, SO_3H, PO_4H_2, N(NO)(OH), SO_2NH_2, P(O)(OH)(NH_2) or tetrazolyl; or a pharmaceutically acceptable salt thereof; and

(b) administering to said patient an amount of a compound of formula (I) effective to inhibit said cells or to sensitize said cells to inhibition by administration of a chemotherapeutic agent.

25. The method of claim 24 wherein Y-Z is a pyridylalkyl ester, a morpholinoalkyl ester, an aminoalkyl ester or a hydroxyalkyl ester.

26. The method of claim 24 wherein Y-Z is a glucamine ester or N-(C_1-C_4)alkyl-glucamine ester of CH_2CO_2H.

27. The method of claim 24 wherein Y-Z is the 1′-D-glucuronate ester of CH_2CO_2H.

28. The method of claim 24 wherein Y-Z is a water-soluble amide of CH_2CO_2H.

29. The method of claim 28 wherein Y-Z is an amino acid amide of CH_2CO_2H.

30. The method of claim 24 wherein the level of PPAR-γ is evaluated.

31. A method for determining the ability of a test agent to inhibit prostate cancer cells comprising contacting a population of cells from a prostate cancer cell line that expresses PPAR-γ with said agent and determining whether the agent increases the expression of PPAR-γ in said cells.

32. A method for determining the ability of a test agent to inhibit prostate cancer cells comprising contacting a population of cells from a prostate cancer cell line that expresses Mcl-1 or Bag-1 with said agent and determining whether the agent decreases the expression of Mcl-1 in said cells.

33. A method for determining the ability of a test agent to inhibit cancer comprising determining whether or not the agent competitively inhibits the receptor-mediated binding of radiolabeled etodolac to cancer cells.

34. The method of claim 33 wherein the cancer cells are etodolac sensitive.

35. The method of claim 33 wherein the etodolac is R(–)-etodolac.

36. The method of claim 31, 32, 33, 34 or 35 further comprising determining whether or not the agent increases the uptake of calcium by cancer cells.

37. The method of claim 36 further comprising determining whether or not the test agent can induce a chemokinetic response in a population of lymphocytes.

38. The method of claim 37 wherein the response enhances the ability of the lymphocytes to exhibit chemotaxis.

39. The method of claim 38 wherein the lymphocytes comprise B-CLL lymphocytes.

40. The method of claim 37 further comprising determining whether or not the test agent can induce apoptosis in cancer cells.

41. The method of claim 40 wherein the cancer cells are CLL cells.

42. The method of claim 40 comprising determining whether or not the test agent can increase caspase-3 activity.

43. The method of claim 40 further comprising determining whether or not the test agent lowers the white cell count of a test animal.

44. The method of claim 43 wherein the test animal is a mouse.

45. The method of claim 43 further comprising determining whether or not the test agent can inhibit cancer induction in the pristane-induced murine MLL model.

46. The method of claim 43 further comprising determining whether or not the test compound can inhibit cancer in the transgenic adenocarcinoma mouse prostate cancer model.

47. A method for determining the susceptibility of prostate cancer to treatment by a compound that activates PPAR-γ expression in prostate cancer cells comprising:

(a) isolating prostate cancer cells from a human subject; and

(b) evaluating whether or not said cells express PPAR-γ at a level sufficient to render them subject to inhibition by said compound.

48. A method for determining the susceptibility of prostate cancer to treatment by a compound thereof that downregulates Mcl-1 or Bag-1 expression in prostate cancer cells comprising:

(a) isolating prostate cancer cells from a human subject;

(b) evaluating whether or not said cells express Mcl-1 at a level sufficient to render them subject to inhibition by said compound.

add C1

add E3